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DATE: Wednesday, July 05, 2006

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☐	L3	BEGLEY adj COLIN	3
☐	L2	NANDURKAR adj HARSHAL	2
☐	L1	ROBB adj LORRAINE	4

END OF SEARCH HISTORY

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 NEWS  5  MAY 10   CA/CAplus enhanced with 1900-1906 U.S. patent records
 NEWS  6  MAY 11   KOREAPAT updates resume
 NEWS  7  MAY 19   Derwent World Patents Index to be reloaded and enhanced
 NEWS  8  MAY 30   IPC 8 Rolled-up Core codes added to CA/CAplus and
                   USPATFULL/USPAT2
 NEWS  9  MAY 30   The F-Term thesaurus is now available in CA/CAplus
 NEWS 10  JUN 02   The first reclassification of IPC codes now complete in
                   INPADOC
 NEWS 11  JUN 26   TULSA/TULSA2 reloaded and enhanced with new search and
                   and display fields

 NEWS EXPRESS      FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
                   CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
                   AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.
                   V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
                   http://download.cas.org/express/v8.0-Discover/

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                                                       ENTRY    SESSION
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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.06       0.27

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=> file medline embase biosis caplus
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.48

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FILE 'EMBASE' ENTERED AT 15:31:15 ON 27 JUN 2006
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FILE 'BIOSIS' ENTERED AT 15:31:15 ON 27 JUN 2006
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FILE 'CAPLUS' ENTERED AT 15:31:15 ON 27 JUN 2006
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=> s interleukin(w)11(w)receptor(w)alpha or il(w)11r(w)alpha
L1          213 INTERLEUKIN(W) 11(W) RECEPTOR(W) ALPHA OR IL(W) 11R(W) ALPHA

=> s l1 and (interleukin(w)11 or il(w)11)
L2          212 L1 AND (INTERLEUKIN(W) 11 OR IL(W) 11)

=> s l2 and interact?
L3           27 L2 AND INTERACT?

=> dup rem
ENTER L# LIST OR (END):l3
PROCESSING COMPLETED FOR L3
L4            14 DUP REM L3 (13 DUPLICATES REMOVED)

=> dis ibib abs l4 1-14

L4   ANSWER 1 OF 14  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2006:333329  CAPLUS
DOCUMENT NUMBER:          144:329305
TITLE:                    Gene expression profiles and predictive model for
                          atherosclerosis and susceptibility to atherosclerosis
INVENTOR(S):              West, Mike; Nevins, Joseph R.; Goldschmidt, Pascal
PATENT ASSIGNEE(S):       Duke University, USA
SOURCE:                   PCT Int. Appl., 279 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     WO 2006026074         A2    20060309    WO 2005-US27989         20050804
     WO 2006026074         A3    20060601
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
```

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2004-651462P P 20040804

AB Genes whose expression is correlated with and determinant of an atherosclerotic phenotype or susceptibility to an atherosclerotic phenotype are provided. Also provided are methods of using the subject atherosclerotic determinant genes or the atherosclerotic susceptibility genes in diagnosis and treatment methods, as well as drug screening methods. Gene expression data from different sections of aorta were analyzed to identify genes and "metagenes" indicative of the susceptibility of vascular tissue to becoming atherosclerotic, or of the mammal from which the vascular sample was derived of developing atherosclerosis. RNA targets isolated from thoracic aorta were hybridized to U95Av2 Affymetrix microarray and processed with the GENECHIP system. A predictive statistical tree model and gene prioritization process identified a set of 208 genes whose expression patterns provide the power to discriminate and predict disease states in the sorta samples. The predictive model correctly classifies 93.5% of the aortic sections as minimally or severely diseased based solely upon their gene expression profiles. Also provided are methods of determining whether a gene is correlated with a disease phenotype, where correlation is determined using at least one parameter that is not expression level and is preferably determined using a binary prediction tree anal.

L4 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:138942 CAPLUS
DOCUMENT NUMBER: 144:211126
TITLE: Osteoporosis treatment with anti-IL-11 antibody, which inhibits formation of a tertiary complex (IL-11, IL-11 α chain, and gp130)
INVENTOR(S): Shaughnessy, Stephen; Austin, Richard Carl
PATENT ASSIGNEE(S): Can.
SOURCE: U.S., 27 pp., Cont.-in-part of U.S. Ser. No. 314,152, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6998123	B1	20060214	US 2000-491982	20000127
CA 2237915	AA	19991119	CA 1998-2237915	19980519
PRIORITY APPLN. INFO.:			CA 1998-2237915 A	19980519
			US 1999-314152 B2	19990519

AB A process is disclosed to treat or alleviate the symptoms of pathol. conditions in which bone d. is decreased, which comprises using antibodies to inhibit, in a patient suffering from such a condition, the formation in vivo of a tertiary complex of interleukin 11 (IL-11), its membrane receptor, and the cell surface glycoprotein gp130. Examples of other such substances are recombinant soluble IL-11 receptor mutants modified, as compared with

native IL-11 receptor, at their gp130 binding site, and peptides which can interact with IL-11. The process of the invention not only inhibits bone resorption and hence bone loss, but also increases the process of bone formation to increase bone d. In one experiment IL-11-neutralizing antibodies halted and even reversed the bone loss in ovariectomized mice. Thus, inhibition of IL-11 biol. activity leads to promotion of new bone formation, bone loss reversal, and increase in bone d. in ovariectomized animals. In vitro results with an IL-11 receptor mutant (H289 to Y289) showed that it was capable of inhibiting IL-11-induced osteoclast formation. Finally, the inventors created a short peptide sequence (antagonist peptide) capable of inhibiting the interaction between IL-11 and IL-11 receptor , a seq. which is homologous to a region in the IL-11 receptor which appears to bind IL -11.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:638674 CAPLUS
DOCUMENT NUMBER: 143:146665
TITLE: Compositions and methods of use of targeting peptides for diagnosis and therapy
INVENTOR(S): Pasqualini, Renata; Arap, Wadih; Kolonin, Mikhail; Zurita, Amado J.
PATENT ASSIGNEE(S): Board of Regents, the University of Texas System, USA
SOURCE: PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2005065418	A2	20050721	WO 2004-US44075		20041230
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2005191294	A1	20050901	US 2004-26999		20041230
PRIORITY APPLN. INFO.:			US 2003-533650P	P	20031231

AB The compns. and methods include targeting peptides selective for tissue selective binding, particularly prostate and/or bone cancer, or adipose tissue. The methods may comprise targeting peptides that bind, for example, cell surface GRP78, IL-11R.alpha. in blood vessels of bone, or prohibitin of adipose vascular tissue. These peptides may be used to induce targeted apoptosis in the presence or absence of at least one pro-apoptotic peptide. Antibodies against such targeting peptides, the targeting peptides, or their mimeotopes may be used for detection, diagnosis and/or staging of a condition, such as prostate cancer or metastatic prostate cancer. Targeting peptide-pro-apoptotic peptide, CGRRAGGSC-GG-D(KLAKLAK)2, bound specifically to IL-11R.alpha. and induced apoptosis in IL-11R.alpha.-pos. prostate cancer cell lines. It was also shown that ligand peptides to GRP78 (i)

target prostate cancer cells in vitro, [ii] home to prostate cancer-derived xenografts in vivo, (iii) bind to human prostate cancer bone metastases and, when coupled to a pro-apoptotic peptide (iv) induce programmed cell death and (v) prevent tumor growth in a human prostate cancer xenograft. A peptide targeting prohibitin, when coupled with the pro-apoptotic peptide, not only prevented obesity development, but also caused a rapid decrease in white fat mass and obesity reversal.

L4 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:34885 CAPLUS
DOCUMENT NUMBER: 142:130333
TITLE: Isolation, culture, characterization and therapeutic use of postpartum cells derived from human umbilical cord
INVENTOR(S): Mistry, Sanjay; Kihm, Anthony J.; Harris, Ian Ross; Harmon, Alexander M.; Messina, Darin J.; Seyda, Agnieszka; Yi, Chin-Feng; Gosiewska, Anna
PATENT ASSIGNEE(S): Ethicon, Incorporated, USA
SOURCE: PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005003334	A2	20050113	WO 2004-US20931	20040625
WO 2005003334	A3	20050407		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004254616	A1	20050113	AU 2004-254616	20040625
CA 2530533	AA	20050113	CA 2004-2530533	20040625
US 2005019865	A1	20050127	US 2004-876998	20040625
US 2005032209	A1	20050210	US 2004-877269	20040625
US 2005037491	A1	20050217	US 2004-877541	20040625
US 2005054098	A1	20050310	US 2004-877012	20040625
US 2005058629	A1	20050317	US 2004-877009	20040625
US 2005058630	A1	20050317	US 2004-877445	20040625
US 2005058631	A1	20050317	US 2004-877446	20040625
AU 2004281371	A1	20050428	AU 2004-281371	20040625
CA 2530412	AA	20050428	CA 2004-2530412	20040625
WO 2005038012	A2	20050428	WO 2004-US20958	20040625
WO 2005038012	A3	20050915		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

EP 1641913 A2 20060405 EP 2004-756395 20040625
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
EP 1649013 A2 20060426 EP 2004-809466 20040625
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
PRIORITY APPLN. INFO.: US 2003-483264P P 20030627
WO 2004-US20931 W 20040625
WO 2004-US20958 W 20040625

AB Cells derived from human umbilical cords are disclosed along with methods for their therapeutic use (such as transplantation). Isolation techniques, culture methods and detailed characterization of the cells with respect to their cell surface markers, gene expression, and their secretion of trophic factors are described.

L4 ANSWER 5 OF 14 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 1

ACCESSION NUMBER: 2005280009 EMBASE
TITLE: **Interleukin-11** receptor signaling is required for normal bone remodeling.
AUTHOR: Sims N.A.; Jenkins B.J.; Nakamura A.; Quinn J.M.W.; Li R.; Gillespie M.T.; Ernst M.; Robb L.; Martin T.J.
CORPORATE SOURCE: Dr. N.A. Sims, Department of Medicine, St. Vincent's Hospital, 41 Victoria Pde, Fitzroy, Vic. 3065, Australia. nsims@medstv.unimelb.edu.au
SOURCE: Journal of Bone and Mineral Research, (2005) Vol. 20, No. 7, pp. 1093-1102. .
Refs: 42
ISSN: 0884-0431 CODEN: JBMREJ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
021 Developmental Biology and Teratology
029 Clinical Biochemistry
033 Orthopedic Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005

AB IL-6 and -11 regulate bone turnover and have been implicated in estrogen deficiency-related bone loss. In this study, deletion of **IL-11** signaling, but not that of IL-6, suppressed osteoclast differentiation, resulting in high trabecular bone volume and reduced bone formation. Furthermore, **IL-11** signaling was not required for the effects of estradiol or estrogen deficiency on the mouse skeleton. Introduction: Interleukin (IL)-6 and -11 stimulate osteoclastogenesis and bone formation in vitro and have been implicated in bone loss in estrogen deficiency. Because of their common use of the gp130 co-receptor signaling subunit, the roles of these two cytokines are linked, and each may compensate for the absence of the other to maintain trabecular bone volume and bone cell differentiation. Materials and Methods: To determine the **interactions** in bone between **IL-11** and IL-6 in vivo and whether **IL-11** is required for normal bone turnover, we examined the bone phenotype of mature male and female **IL-11** receptor knockout mice (**IL-11R.alpha**.1(-/-)) and compared with the bone phenotype of IL-6(-/-) mice and mice lacking both IL-6 and **IL-11R.alpha**.. To determine whether **IL-11** is required for the effects of estrogen on trabecular bone, mature **IL-11R.alpha**.1(-/-) mice were ovariectomized and treated with estradiol. Results: In both male and female **IL-11R.alpha**.1(-/-) mice, trabecular bone volume was significantly higher than that of wildtype controls. This was associated with low bone resorption and low

bone formation, and the low osteoclast number generated by IL-11R.alpha.1(-/-) precursors was reproduced in ex vivo cultures, whereas elevated osteoblast generation was not. Neither trabecular bone volume nor bone turnover was altered in IL-6(-/-) mice, and compound IL-6(-/-):IL-11R.alpha.1(-/-) mice showed an identical bone phenotype to IL-11R.alpha.1(-/-) mice. The responses of IL-11R.alpha.1(-/-) mice to ovariectomy and estradiol treatment were the same as those observed in wildtype mice. Conclusions: IL-11 signaling is clearly required for normal bone turnover and normal trabecular bone mass, yet not for the effects of estradiol or estrogen deficiency on the skeleton. In the absence of IL-11R.alpha., increased trabecular bone mass seems to result from a cell lineage-autonomous reduction in osteoclast differentiation, suggesting a direct effect of IL-11 on osteoclast precursors. The effects of IL-11R.alpha. deletion on the skeleton are not mediated or compensated for by changes in IL-6 signaling. .COPYRGT. 2005 American Society for Bone and Mineral Research.

L4 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:66610 CAPLUS
DOCUMENT NUMBER: 142:368954
TITLE: Identification of novel TCDD-regulated genes by microarray analysis
AUTHOR(S): Hanlon, Paul R.; Zheng, Wenchao; Ko, Alex Y.; Jefcoate, Colin R.
CORPORATE SOURCE: Molecular and Environmental Toxicology Center, University of Wisconsin-Madison, WI, 53706, USA
SOURCE: Toxicology and Applied Pharmacology (2005), 202(3), 215-228
CODEN: TXAPA9; ISSN: 0041-008X
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB TCDD exposure of multipotential C3H10T1/2 fibroblasts for 72 h altered the expression of over 1000 genes, including coordinated changes across large functionally similar gene clusters. TCDD coordinately induced 23 cell cycle-related genes similar to epidermal growth factor (EGF)-induced levels but without any affect on the major mitogenic signaling pathway (extracellular signal-regulated kinase, ERK). TCDD treatment also decreased glycolytic and ribosomal clusters. Most of these TCDD-induced changes were attenuated by the presence of EGF or an adipogenic stimulus, each added during the final 24 h. TCDD prevented 10% of EGF-induced gene responses and 40% of adipogenic responses. Over 100 other genes responded to TCDD during adipogenesis. This group of responses included complete suppression of three proliferins and stimulations of several cytokine receptors. Despite these varied secondary effects of TCDD, direct AhR activation measured by integrated AhR-responsive luciferase reporters was similar under quiescent, EGF-stimulated or adipogenic conditions. Only 23 genes were similarly induced by TCDD regardless of conditions and 10 were suppressed. These 23 genes include: 4 genes previously recognized to contain AhR response elements (cytochrome P 450 (CYP) 1B1, CYP1A1, NAD(P)H quinone reductase 1 (NQO1), and aldehyde dehydrogenase 3A1); two novel oxidative genes (alc. dehydrogenase 3 and superoxide dismutase 3); and glypican 1, a plasma membrane proteoglycan that affects cell signaling. Further expts. demonstrated that TCDD maximally induced NQO1, glypican 1 and alc. dehydrogenase 3 by 6 h. Glypican 1 activates the actions of many growth factors and therefore may contribute to secondary effects on gene expression.
REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 14 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:171839 BIOSIS
DOCUMENT NUMBER: PREV200300171839
TITLE: Expression and function of **interleukin-11** and its receptor alpha in the human endometrium.
AUTHOR(S): Karpovich, Natalia; Chobotova, Katya; Carver, Janet; Heath, John K.; Barlow, David H.; Mardon, Helen J. [Reprint Author]
CORPORATE SOURCE: Department of Obstretrics and Gynaecology, University of Oxford, John Radcliffe Hospital, Women's Centre, Level 3, Oxford, OX3 9DU, UK
helen.mardon@obs-gyn.ox.ac.uk
SOURCE: Molecular Human Reproduction, (February 2003) Vol. 9, No. 2, pp. 75-80. print.
ISSN: 1360-9947 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Apr 2003
Last Updated on STN: 2 Apr 2003

AB The **interleukin-11 (IL-11)** receptor alpha has an important function in decidualization of mouse endometrial stroma but the function of IL-11 and its receptor in the human endometrium remains unknown. The mRNA for IL-11 and its receptor alpha in human endometrial tissue samples were analysed by semi-quantitative RT-PCR and RNase protection assays respectively. The proteins were detected in frozen endometrial tissue samples by immunofluorescence. The effect of heparin-binding epidermal growth factor (HB-EGF) on secretion of **IL-11** by cultured endometrial stromal cells was assessed by enzyme-linked immunosorbent assay. The proliferative potential of **IL-11** in endometrial stromal cells was assessed by (3H)thymidine uptake. **IL-11** and its receptor alpha mRNAs and proteins were detected in the endometrium throughout the cycle. Distinct patterns of localization of the ligand and receptor were observed. HB-EGF induced **IL-11** secretion by cultured stromal cells, and **IL-11** induced (3H)thymidine uptake by these cells. Our data suggest that **IL-11-receptor interactions** may perform different functions in the human endometrium at different stages of the cycle, and that secretion of **IL-11** is modulated by local growth factors.

L4 ANSWER 8 OF 14 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN DUPLICATE 2
ACCESSION NUMBER: 2003413458 EMBASE
TITLE: Characterization of a potent human **interleukin-11** agonist.
AUTHOR: Harmegnies D.; Wang X.-M.; Vandenbussche P.; Leon A.; Vusio P.; Grotzinger J.; Jacques Y.; Goormaghtigh E.; Devreese B.; Content J.
CORPORATE SOURCE: J. Content, Institut Pasteur de Bruxelles, rue Engeland 642, B-1180 Brussels, Belgium. jcontent@pasteur.be
SOURCE: Biochemical Journal, (1 Oct 2003) Vol. 375, No. 1, pp. 23-32. .
Refs: 59
ISSN: 0264-6021 CODEN: BIJOAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Oct 2003
Last Updated on STN: 30 Oct 2003

AB Human **interleukin-11** (hIL-11) is a multi-potential cytokine that is involved in numerous biological activities, such as

haematopoiesis, osteoclastogenesis, neurogenesis and female fertility, and also displays anti-inflammatory properties. **IL-11** is used clinically to treat chemotherapy-induced thrombocytopenia. Because of its broad spectrum of action, improved **IL-11** agonists, as well as **IL-11** antagonists, could be of interest for numerous clinical applications. **IL-11** signalling is dependent on the formation of a tripartite ligand-receptor complex consisting of **IL-11**, the IL-11R (**IL-11** receptor) α subunit (responsible for the specificity of the **interaction**) and gp130 (glycoprotein 130) receptor β subunit (responsible for signal transduction). The **interaction** between **IL-11** and **IL-11R**. **alpha**. subunit occurs at its recently assigned site I. We have designed an **IL-11** mutein whose hydrophobicity at site I has been increased. The mutein has been characterized in terms of structure, affinity, specificity and bioactivity. Electrophoretic analysis, gel filtration, IR spectroscopy and CD indicate that this new protein is more compact than wild-type **IL-11**. It binds to **IL-11R**.**alpha**. with a three-fold-enhanced affinity, and retains the ability to recruit gp130 through site II. However, analysis of its biological activity revealed a complex pattern: although this mutein is 60-400-fold more active than wild-type **IL-11** on the proliferation of 7TD1 murine hybridoma cell, it is less active than **IL-11** on the proliferation of B9 cells, another murine hybridoma cell line. The results are interpreted on the basis of an **IL-11** conformational change induced by the mutations, and the preferential use by the mutein of another unknown transducing receptor chain.

L4 ANSWER 9 OF 14 EMBASE DUPLICATE 3

ACCESSION NUMBER: 2002308748 EMBASE
TITLE: Transcriptional program of mouse osteoclast differentiation governed by the macrophage colony-stimulating factor and the ligand for the receptor activator of NFκB.
AUTHOR: Cappellen D.; Luong-Nguyen N.-H.; Bongiovanni S.; Grenet O.; Wanke C.; Susa M.
CORPORATE SOURCE: M. Susa, Novartis Pharma Research, Arthr./Bone Metab. Therapeutic Area, WKL-125.9.12, CH-4002 Basel, Switzerland. mira.susa_spring@pharma.novartis.com
SOURCE: Journal of Biological Chemistry, (14 Jun 2002) Vol. 277, No. 24, pp. 21971-21982. .
Refs: 61
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 26 Sep 2002
Last Updated on STN: 26 Sep 2002

AB Cytokines macrophage colony stimulating factor (M-CSF) and the receptor activator of NFκB ligand (RANKL) induce differentiation of bone marrow hematopoietic precursor cells into bone-resorbing osteoclasts without the requirement for stromal cells of mesenchymal origin. We used this recently described mouse cell system and oligonucleotide microarrays representing about 9,400 different genes to analyze gene expression in hematopoietic cells undergoing differentiation to osteoclasts. The ability of microarrays to detect the genes of interest was validated by showing expression and expected regulation of several osteoclast marker genes. In total 750 known transcripts were up-regulated by ≥2-fold, and 91% of them at an early time in culture, suggesting that almost the whole differentiation program is defined already in pre-osteoclasts. As expected, M-CSF alone induced the receptor for RANKL

(RANK), but also, unexpectedly, other RANK/NFκB pathway components (TRAF2A, PI3-kinase, MEKK3, RIPK1), providing a molecular explanation for the synergy of M-CSF and RANKL. Furthermore, interleukins, interferons, and their receptors (IL-1α, IL-18, IFN-β, IL-11R.alpha.2, IL-6/11R gp130, IFNγR) were induced by M-CSF. Although interleukins are thought to regulate osteoclasts via modulation of M-CSF and RANKL expression in stromal cells, we showed that a mix of IL-1, IL-6, and IL-11 directly increased the activity of osteoclasts by 8.5-fold. RANKL induced about 70 novel target genes, including chemokines and growth factors (RANTES (regulated on activation, normal T cell expressed and secreted), PDGFα, IGF1), histamine, and α1A-adrenergic receptors, and three waves of distinct receptors, transcription factors, and signaling molecules. In conclusion, M-CSF induced genes necessary for a direct response to RANKL and interleukins, while RANKL directed a three-stage differentiation program and induced genes for interaction with osteoblasts and immune and nerve cells. Thus, global gene expression suggests a more dynamic role of osteoclasts in bone physiology than previously anticipated.

L4 ANSWER 10 OF 14 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2001224308 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11177577
TITLE: Interleukin-11 modulates Th1/Th2 cytokine production from activated CD4+ T cells.
AUTHOR: Bozza M; Bliss J L; Dorner A J; Trepicchio W L
CORPORATE SOURCE: Department of Molecular Medicine, Genetics Institute, Andover, MA 01810, USA.
SOURCE: Journal of interferon & cytokine research : the official journal of the International Society for Interferon and Cytokine Research, (2001 Jan) Vol. 21, No. 1, pp. 21-30. Journal code: 9507088. ISSN: 1079-9907.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 2 May 2001
Last Updated on STN: 2 May 2001
Entered Medline: 26 Apr 2001

AB Recombinant human interleukin-11 (rHuIL-11) is a pleiotropic cytokine with effects on multiple cell types. rHuIL-11 reduces activated macrophage activity and downregulates production of proinflammatory mediators, such as tumor necrosis factor-alpha (TNF-alpha) and nitric oxide (NO). In vitro and in vivo, rHuIL-11 inhibits production of key immunostimulatory cytokines, including IL-12 and interferon-gamma (IFN-gamma). rHuIL-11 has recently demonstrated immunomodulatory activity to downregulate IFN-gamma production, increase IL-4 production, and reduce inflammatory tissue injury in a human psoriasis clinical trial. The cellular mechanisms of these effects are not fully elucidated. We demonstrate here that expression of gp130 and IL-11 receptor (IL-11R) alpha mRNA, components of the IL-11R complex, are detected in human and murine CD4(+) and CD8(+) lymphocytes, suggesting that rHuIL-11 can directly interact with T cells. In a cell culture model of murine T cell differentiation, rHuIL-11 acts to inhibit IL-2 production as well as IL-12-induced IFN-gamma production and enhances IL-4 and IL-10 production. rHuIL-11 had no effect on T cell proliferation. The ability of rHuIL-11 to modulate cytokine production from activated CD4(+) T cells provides a mechanism through which rHuIL-11 may ameliorate such inflammatory diseases as psoriasis.

L4 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:479428 CAPLUS
DOCUMENT NUMBER: 129:104682

TITLE: Methods for modulating fertility and the maintenance of pregnancy using IL-11
INVENTOR(S): Robb, Lorraine Grace; Nandurkar, Harshal Hanumant; Begley, Colin Glenn
PATENT ASSIGNEE(S): Amrad Operations Pty Ltd, Australia
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9827996	A1	19980702	WO 1997-AU880	19971224
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9878715	A1	19980717	AU 1998-78715	19971224
AU 739063	B2	20011004		
EP 956039	A1	19991117	EP 1997-948654	19971224
R: DE, FR, GB, IT				
US 6669934	B1	20031230	US 1999-331569	19990827
AU 762879	B2	20030710	AU 2002-10046	20020104
US 2004043000	A1	20040304	US 2003-659200	20030910

PRIORITY APPLN. INFO.:

AU 1996-4393	A	19961224
AU 1998-78715	A3	19971224
WO 1997-AU880	W	19971224
US 1999-331569	A1	19990827

AB The present invention relates generally to a method for controlling fertility and/or modulating the maintenance of pregnancy in animals using **interleukin-11** or a functional derivative or homolog thereof or an effective amount of an agonists or antagonist of the **interaction** between **IL-11** and **IL-11R.alpha.**. The method can also involve modulating the levels of expression of the gene encoding **IL-11** or **IL-11R.alpha.**. **IL-11** or an agonist of **IL-11** can be co-administered with cytokines, selected from LIF, CNTF, IL-6, and OSM or functional derivs. or homologs thereof. The animals of the invention include humans, primates, livestock, companion animals, laboratory test animals, of captive wild animals. The present invention further provides an animal model (comprising a mutation in at least one allele for **IL-11** and/or **IL-11R.alpha.**) useful for screening for therapeutic agents to treat infertility, to prevent or reduce spontaneous abortion and/or as contraceptive agents in animals.
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 12 OF 14 EMBASE DUPLICATE 5
ACCESSION NUMBER: 1998252864 EMBASE
TITLE: Maternal **IL-11R.alpha.** function is required for normal decidua and fetoplacental development in mice.
AUTHOR: Bilinski P.; Roopenian D.; Gossler A.
CORPORATE SOURCE: P. Bilinski, Institut fur Genetik, Heinrich-Heine Univ. Dusseldorf, 40225 Dusseldorf, Germany. ago@aretha.jax.org
SOURCE: Genes and Development, (15 Jul 1998) Vol. 12, No. 14, pp.

2234-2243. .
Refs: 34
ISSN: 0890-9369 CODEN: GEDEEP
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 010 Obstetrics and Gynecology
021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 14 Aug 1998
Last Updated on STN: 14 Aug 1998

AB In eutherian mammals, implantation and establishment of the chorioallantoic placenta are essential for embryo development and survival. As a maternal response to implantation, uterine stromal cells proliferate, differentiate, and generate the decidua, which encapsulates the conceptus and forms the maternal part of the placenta. Little is known about decidual functions and the molecular **interactions** that regulate its development and maintenance. Here we show that the receptor for the cytokine **interleukin-11** (**IL-11R.alpha.**) is required specifically for normal establishment of the decidua. Females homozygous for a hypomorphic **IL-11R.alpha.** allele are fertile and their blastocysts implant and elicit the decidual response. Because of reduced cell proliferation, however, only small deciduae form. Mutant deciduae degenerate progressively, and consequently embryo-derived trophoblast cells generate a network of trophoblast giant cells but fail to form a chorioallantoic placenta, indicating that the decidua is essential for normal fetoplacentation. **IL-11R.alpha.** is expressed in the decidua as well as in numerous other tissues and cell types, including the ovary and lymphocytes. The differentiation state and proliferative responses of B and T-lymphocytes in mutant females were normal, and wild-type females carrying **IL-11R.alpha.** mutant ovaries had normal deciduae, suggesting that the decidualization defects do not arise secondarily as a consequence of perturbed **IL-11R.alpha.** signaling defects in lymphoid organs or in the ovary. Therefore, **IL-11R.alpha.** signaling at the implantation site appears to be required for decidua development.

L4 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:342229 CAPLUS
DOCUMENT NUMBER: 125:8495
TITLE: Cloning of cDNA for novel hemopoietin receptors of mammals
INVENTOR(S): Hilton, Douglas James
PATENT ASSIGNEE(S): Amrad Operations Pty. Ltd., Australia
SOURCE: PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9607737	A1	19960314	WO 1995-AU578	19950905

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9534652	A1	19960327	AU 1995-34652		19950905
AU 690743	B2	19980430			
EP 804576	A1	19971105	EP 1995-931079		19950905
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE					
JP 10505068	T2	19980519	JP 1995-509002		19950905
CA 2197873	AA	19960314	CA 1995-2197873		19950909
US 6274708	B1	20010814	US 1996-702665		19961220
US 7002000	B1	20060221	US 2000-532263		20000322
US 2003149236	A1	20030807	US 2001-853105		20010510
US 2006051842	A1	20060309	US 2005-204563		20050816
PRIORITY APPLN. INFO.:			AU 1994-7901	A	19940905
			AU 1994-7902	A	19940905
			WO 1995-AU578	W	19950905
			US 1996-702665	A3	19961220
			US 2000-532263	A1	20000322

AB The cDNA encoding α-chain of **interleukin 11** are isolated from mouse and human and their amino acid sequences deduced. The novel receptors contain a 5-amino-acid motif, WSXWS. The receptor mols. or components or parts thereof and their genetic sequences of the present invention are useful in the development of a wide range of agonists, antagonists and therapeutics and diagnostic reagents based on ligand **interaction** with its receptor.

L4 ANSWER 14 OF 14 MEDLINE on STN DUPLICATE 6

ACCESSION NUMBER: 96226094 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8637716
TITLE: The human **IL-11** receptor requires gp130 for signalling: demonstration by molecular cloning of the receptor.
AUTHOR: Nandurkar H H; Hilton D J; Nathan P; Willson T; Nicola N; Begley C G
CORPORATE SOURCE: Walter and Eliza Hall Institute of Medical Research, Victoria, Australia.
CONTRACT NUMBER: CA 22556 (NCI)
SOURCE: Oncogene, (1996 Feb 1) Vol. 12, No. 3, pp. 585-93. Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19 Jul 1996
Last Updated on STN: 3 Feb 1997
Entered Medline: 5 Jul 1996

AB We describe the molecular cloning of a cDNA for the alpha chain of the human **IL-11** receptor (**IL-11R alpha**) and demonstrate the requirement of either the human or mouse gp130 molecule for signalling. cDNA clones encoding **IL-11R alpha** were isolated from a bone marrow cDNA library using a fragment from the murine **IL-11R alpha** as a probe. The human receptor was predicted to consist of 422 amino acids and was found to share 84% identity with the murine protein. In the extra-cellular region it exhibited a single hemopoietin domain with conserved cysteine residues and WSTWS motif. The transmembrane region was followed by a short cytoplasmic tail which did not contain a tyrosine kinase domain. **Interaction** of the human **IL-11R alpha** with murine gp130 was demonstrated: expression of the human **IL-11R alpha** in murine M1 cells which constitutively express murine gp130 (and murine LIF receptor), resulted in the generation of specific high-affinity binding sites for **IL-11** (Kd = 250 pM). In addition, expression of the human **IL-11R alpha** in these cells permitted the induction of macrophage differentiation in response to **IL-11**. These results suggested that the human **IL-**

11R alpha chain was able to form a functional receptor complex in association with murine gp130. The requirement of gp130 for signalling was confirmed by expression of the human IL-11R alpha in Ba/F3 cells. BaF3 cells that expressed the human IL-11R alpha alone showed binding of radiolabelled IL-11 but no proliferative response. Introduction of human gp130 into these cells resulted in high-affinity IL-11 binding sites and IL-11 dependent cellular proliferation. Thus these results demonstrated the absolute requirement of gp130 for signalling.

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FULL ESTIMATED COST	62.66	63.14

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jun 23, 2006 (20060623/UP).